

seen presenting subarachnoid hæmorrhage, associated with a recent membrane, where it has been possible to trace the actual rupture in the arachnoid through which the blood escaped. There is a greater tendency for the blood to rupture direct into the sub-dural space than to spread beneath the arachnoid, which is accounted for by the conditions present in many cases of insanity and general paralysis, in which the adhesion between pia and arachnoid, over the summits of the gyri, is so great as to constitute one membrane, and in bursting into the arachnoid cavity, the blood follows the lines of least resistance. These pachymeningitic membranes occur under conditions which favor hæmorrhage, being associated in overwhelming proportion with brain degeneration and atrophy, especially when these conditions are combined with congestion of the meninges. Brain atrophy is the proximate cause of the hæmorrhage, the meningeal vessels suffering thereby a loss of support, and with this we have meningeal congestion, and further, degeneration of the vessels themselves, a series of circumstances highly favorable to the effusion of blood. A. F.

PROGRESSIVE BULBAR PARALYSIS.

Nothnagel (*Internat. klin. Rundschau*, 1891, No. 17-20), in presenting a patient with this disease, makes the following observations:

Speech disturbances, such as difficulty in articulation, lead to the diagnosis of bulbar paralysis. The patient speaks as if he had something in his mouth. The mobility of the tongue seems to be interfered with, the lingual sounds giving him the greatest difficulty. There are demonstrable fibrillary contractions and atrophy of the tongue. We have to do with either a peripheral lesion affecting the trunk of the hypoglossal nerve or an atrophy of the hypoglossal nucleus. In lingual paralyzes of central origin there is paresis of the tongue, yet fibrillary contractions and pronounced atrophy are absent. A third possibility is the wasting of the substance of the tongue from disease in the organ itself. The wasting of the tongue in tabes seems to be due to atrophy of the hypoglossal nucleus. The commonest causes of dysarthria are either dementia paralytica or progressive bulbar paralysis. In the former, speech is syllabic, in fact, ataxic. In the present case there is, furthermore, paresis of the facial nerve and the muscles of deglutition. We thus have a case of glosso-labial-pharyngo-laryngeal paralysis. In its further progress there will be paresis in the distribution of the accessorius and the vagus (marked

increase in the frequency of the pulse and dyspnœa). The nuclei situated collectively in the medulla will be attacked one after another. We are dealing with a primary degeneration of the nuclei and not with an inflammation. One speaks of acute apoplectiform bulbar paralysis, when the manifestations appear in an apoplectiform and sudden manner (hæmorrhage or thrombosis in the vertebral). Lepin has, therefore, called attention to the fact, that in bilateral lesion of the lower portion of the central convolutions we have a similar clinical picture (pseudo-bulbar paralysis). However, the paralyzes are not symmetrical, and moreover the paralyzes of the extremities are demonstrable, while the electrical irritability remains normal. The symptoms also appear suddenly. We also differentiate a total from a partial ophthalmoplegia. In the present case the latter condition exists (left ptosis, contracted pupil, paresis of the left external rectus and the right internus).

The prognosis is unfavorable in all degenerative processes affecting the brain and spinal cord. The most rational plan of treatment is by electricity. In most cases when the pharyngeal muscles become atrophied and paretic, we must look after the patient's nutrition. Internally we may administer iodide of potassium or ergotin. Some have seen improvement following the use of nitrate of silver. (*Centralblatt f. klin. Med.*, No. 37, 1891.)

W. M. L.

CLINICAL.

COMPRESSION OF THE CAUDA EQUINA AND ITS TREATMENT.

L. Laguer records the following case: A nineteen-year-old locksmith, L., fell sick in September, 1888, with very violent pains in the small of his back, especially severe during the night and which resisted all treatment. Examination at first revealed neither motor nor sensory disturbances, atrophy, nor alterations of the reflexes. An improvement took place which lasted several months. Finally, in September, 1890, not only the back pains returned, but also disturbances in urination and defecation. The patellar reflex on the right side was absent, on the left was diminished. The recti femoris on both sides were slightly atrophied. The patient would walk about in a shuffling manner, with his body bent forward. Slight kyphosis of the lumbar region developed, as well as a